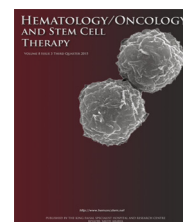




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IMAGES AND DIAGNOSIS

Giant ribbon-like platelets mimicking microfilaria in a *JAK2*-positive myeloproliferative neoplasm



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A 65-year-old man presented with weight loss, easy fatigability, night sweats, and massive splenomegaly, ongoing for 2 months. There was no history of blood transfusions and no lymphadenopathy was detected. Hemoglobin was 125 g/L, total leukocyte count was $6.9 \times 10^9/L$, and platelets were $358 \times 10^9/L$. Blood film showed normocytic normochromic red cells and 85% neutrophils with a mild shift-to-the-left (2% metamyelocytes). A few long ribbon-like eosinophilic fragments were present (Figure 1) that were reminiscent of microfilaria (larval forms of various filarial nematodes) at scanner magnification. However, the oil immersion objective revealed cytoplasmic granularity. This, along with the absence of typical cephalic space and multiple nuclear columns of the nematode larvae, confirmed the structures to be simply extremely elongated platelets. Similar, but shorter, elongated platelets were also otherwise abundant (Figure 1, inset). A literature search revealed that platelet-strings or ribbons are normally shed by megakaryocytes into marrow sinuses and these then fragment into numerous platelets.¹

Our patient's bone marrow was markedly hypercellular with panmyelosis and especially prominent megakaryocytic

hyperplasia and clustering (Figure 2). The megakaryocytes varied from predominantly normal to a few hypolobate and occasional hyperlobate forms (Figure 2). Reticulin was not increased (European Myelofibrosis Network, EUMNET grade, 0–3). Amplification refractory mutation system-polymerase chain reaction revealed heterozygous state for the *JAK2 V617F* mutation (Figure 3). Reverse transcriptase-polymerase chain reaction was negative for the *BCR-ABL1* fusion gene.

Filariasis is endemic in many parts of India² and coexistent malignant and parasitic disorders are well-described.^{3,4} A high index of suspicion is therefore useful for tropical pathologists; however, the possibility of misdiagnosis in the current case was unlikely once close examination was done by experts well-versed in parasite morphology.

This case, apart from the unusual platelet morphology, also illustrates the practical dilemmas faced by hematopathologists in the classification of *JAK2*-positive myeloproliferative neoplasms with overlapping clinical, morphological, and hematological findings. Our patient does not meet blood count criteria for overt polycythemia vera (PV) or essential thrombocythemia. He may fit into primary myelofibrosis (a prefibrotic cellular stage) or a prepolycythemic stage of PV. The large spleen, however, argues against prefibrotic primary myelofibrosis, while

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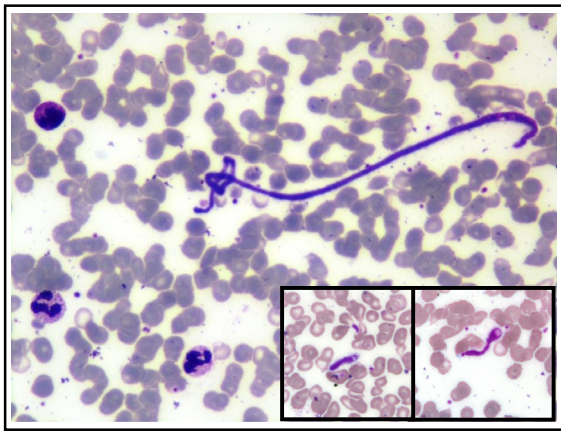


Figure 1 A giant ribbon-like platelet in circulation. (May-Grünwald Giemsa, 400). Several other elongated, albeit shorter platelets were also seen (inset; May-Grünwald Giemsa, 1000).

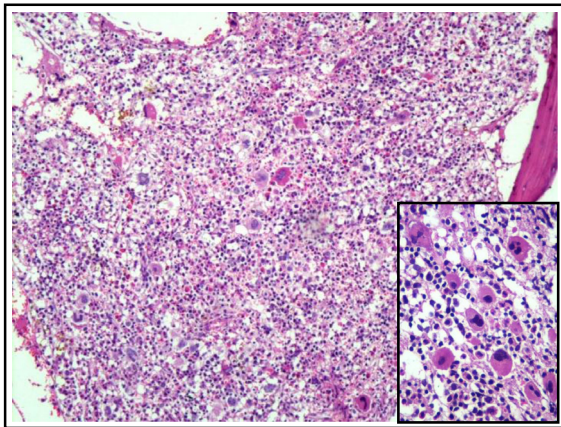


Figure 2 The bone marrow biopsy showed near-maximal cellularity with panmyelosis (hematoxylin and eosin, 100). Megakaryocytic proliferation and nuclear atypia were prominent (inset; hematoxylin and eosin, 400).

the heterozygous state for *JAK2* mutation is less likely in PV. The case was, therefore, finally diagnosed as myeloproliferative neoplasms-unclassifiable; with the suggestion to carefully follow up his blood counts and clinical status.

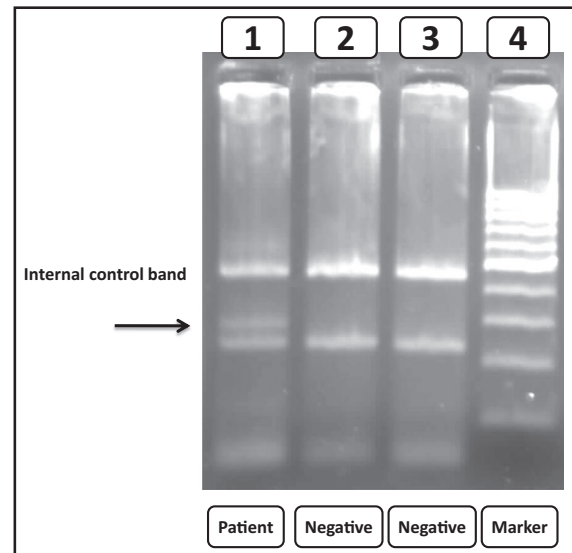


Figure 3 A 2% agarose gel resolving the products of amplification refractory mutation system-polymerase chain reaction for a *JAK2* V617F mutation. The patient data in Lane 1 shows two bands indicating heterozygosity for the mutation. The negative cases in Lanes 2 and 3 do not show the mutant band (arrow).

Conflicts of interest

The authors have no conflicts of interest to declare.

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